Brief Communication

Severe Polyhydramnios Associated with Congenital Hypothyroidism and Hyponatraemia: A Case Report

Mohammad Khassawneh[1] and Zuhair Amarin* [2]

Abstract

This reports a case of congenital hypothyroidism associated with fetal polyhydramnios and severe neonatal hyponatremia. This patient is a premature 32- week infant who had poor oral feeding and severe hyponatremia that persisted till a diagnosis and treatment of hypothyroidism was established at the 37- week corrected age (5 weeks of age). We here attempt to suggest another mechanism of polyhydramnios with hypothyroidism that is not linked to neck hyperextension. The relationship between poor feeding and swallowing in this patient and severe polyhydramnios was discussed. Literature review of the role of thyroid hormone in sodium homeostasis and urine output in relation to polyhydramnios was also discussed. This case report invites further research to understand the role of thyroid hormone in urine output and sodium homeostasis.

Keywords: Polyhydramnios, Congenital hypothyroidism, neonatal hyponatremia.

(J Med J 2008; Vol. 42 (1): 53-55)

Received	***Accepted***
March 26, 2007	*August 2, 2007*

Introduction

Polyhydramnios is usually defined as a deepest vertical pool of the amniotic fluid with the ultrasound of ≥ 8 cm, or an amniotic fluid index above the 95th centile for the gestational age. It complicates between 1 - 3.5% of all pregnancies,[1] and has been shown to be an independent risk factor for perinatal and intrapartum complications among preterm births. [2]

Diabetes mellitus, upper gastrointestinal obstruction, impaired fetal swallowing, fetal polyuria, cardiac failure, fetal anemia and infections may cause Polyhydramnios. Idiopathic polyhydramnios is still blamed for many cases of polyhydramnios. We are presenting in this case report a case of polyhydramnios associated with congenital hypothyroidism and severe hyponatremia. Our attempt is to link these findings in this neonate with the history of polyhydramnios in order to find the cause of this idiopathic polyhydramnios.

1- Department of Pediatrics, Faculty of Medicine, Jordan University of Science and Technology, Irbid.
2- Department of Obstetrics and Gynecology, Faculty of Medicine, Jordan University of Science and Technology, Irbid.
*** Correspondence should be addressed to:**
Mohammad Khassawneh
P.O. Box: 126, Irbid, Aidone, Jordan
E- mail: deema321@yahoo.com

Case Report

A 30-year-old healthy woman booked for antenatal care in her second pregnancy with acute severe polyhydramnios. Her first pregnancy was complicated by intra uterine growth restriction and severe polyhydramnios. It ended with a full-term normal vaginal delivery.

In the current pregnancy, amnio-reduction was performed at the 26th week, and was repeated at the 28th and 30th weeks of gestation. At the 32nd week of gestation, a premature baby boy was born by caesarean section secondary to the repeated needs for amnio-reduction. The birth weight was 1390 grams. The baby suffered from respiratory distress syndrome. His hospital course was complicated by severe hyponatremia (121meq/l), for which he received 8meq/kg/day sodium replacement. Normal physical examination, tolerance of naso-gastric feeding and normal stool pattern ruled out gastrointestinal or neuromuscular causes for the polyhydramnios. At the 5th week of age (37 weeks corrected age) a neck mass was noticed. Thyroid function tests were consistent with neonatal hypothyroidism (TSH of 100 mcu/l and T4 of 5.8 ng/l). Also, Thyroid scan was within the normal limit. L-thyroxin started at 20 mcg per day. Oral intake and weight gain, which were initially very poor, improved dramatically after thyroid hormone replacement. The baby was discharged home at the 6th week of age. At follow-up, there was a weight gain of 45 gram per day and TSH had normalized.

Discussion

Although a large proportion of cases are idiopathic and no obvious cause can be ascertained, severe polyhydramnios associated with goitrous fetal hypothyroidism is not uncommon. The pathogenesis was used to be considered a swallowing problem. [3] Another possible mechanism is impairment of swallowing the amniotic fluid in intrauterine life secondary to hypothyroid state. Our patient was noticed to have a small goiter and hypothyroidism at the 35th day of life (37 weeks corrected gestational age). This is considered a case of congenital hypothyroidism since diagnosis was established before the expected date of delivery. L-thyroxin started at the 37th week of corrected gestational age and was associated with improved oral intake and better weight gain. Resolution of polyhydramnios has been documented in few cases after treatment with intra-amniotic thyroid hormone replacement. [4]

Our baby had severe hyponatremia that responded to sodium replacement. Hyponatremia was described in few cases of adult hypothyroidism but less frequently in cases of congenital hypothyroidism. Asami and Uchiyama demonstrated no causal relationship between sodium level and thyroid hormone level in cases of congenital hypothyroidism that were on treatment. [5] This is different from our finding in this case report although L-thyroxin treatment started at the 35th day of life. The mechanism of hyponatremia could be linked to polyhydramnios since amniotic fluid volume is dependant on fetal urine output rate, however, the renal causes of polyhydramnios such as fetal diabetes insipidus and hyperprostaglandinuric tubular syndrome have been described with it. [6] Perhaps thyroid hormone has an effect on maturation of renal tubular function and control of fetal urine output. Further research in this area is required.

Thyroid hormones, TSH and thyroid binding globulin increase progressively throughout intrauterine life. [7] This increase is altered in growth-restricted children, which indicates a role for this hormone in growth and development during fetal life. The fetus is dependent on its own thyroid hormone. Bearing in mind the association between hypothyroidism and polyhydramnios may be helpful in early diagnosis and treatment of fetal congenital hypothyroidism. Moreover, resolution of polyhydramnios after thyroid hormones replacement may decrease risks associated with polyhydramnios such as preterm labor and placental abruption.

In Conclusion: Association between hypothyroidism and polyhydramnios allows early diagnosis and treatment of cases of fetal hypothyroidism. This decreases the chances of preterm birth due to polyhydramnios, with a subsequent reduction in perinatal mortality and morbidity. Further studies are needed to investigate possible relationship between thyroid function and sodium homeostasis.

References

1. Hill LM, Breckle R, Thomas ML, Fries JK. Polyhydramnios: ultrasonically detected prevalence and neonatal outcome. Obstetrics and Gynecology 1987; 67: 21-25
2. Mazor M, Ghezzi F, Maymon E, et al. Polyhydramnios is an independent risk factor for perinatal mortality and intrapartum morbidity in preterm delivery. European Journal of Obstetrics, Gynecology and Reproductive Biology 1996; 70: 41-47.
3. Agrawal P, Ogilvy-Stuart A, Lees C. Intrauterine diagnosis and management of congenital goitrous hypothyroidism. Ultrasound in Obstetrics and Gynecology 2002; 19: 501-505.
4. Hashimoto H, Hashimoto K, and Suehara N. Successful in-utero treatment of fetal goitrous hypothyroidism: case report and review of the literature. Fetal Diagnosis and Therapy 2006; 21: 360-365.
5. Asami T, Uchiyama M. Sodium handling in congenitally hypothyroid neonates. Acta Paediatrica, 2004; 93: 22-24.
6. Schmitz PH, de Meijer PH, Meinders AE. Hyponatremia due to hypothyroidism: a pure renal mechanism. Netherlands Journal of Medicine 2001; 58: 143-149.
7. Lott JA, Sardoria-Iyer M, Speakman KS, Lee KK. Age-dependent cutoff values in screening newborns for hypothyroidism. Clinical Biochemistry 2004; 37: 791-797.

زيادة السائل الأمنيوسي في حالة نقص خلقي لهرمونات الغدة الدرقية مصاحبة لنقص شديد في مستوى املاح الصوديوم: تقرير عن حالة

محمد خصاونه[1]، زهير عمارين[2]

1- قسم طب الأطفال؛ 2- قسم النسائية والتوليد، كلية الطب، جامعة العلوم والتكنولوجيا الأردنية، اربد، الأردن.

الملخص

تتضمن هذه الدراسة تقريراً عن حالة لنقص خلقي لهرمونات الغدة الدرقية مرتبط بزيادة كبيرة في السائل الأمنيوسي اثناء الحمل وانخفاض شديد في مستوى املاح الصوديوم في الدم. هذا المريض هو طفل وُلد مبكراً بعد حملٍ دام 32 أسبوعاً، وكان يعاني من عدم القدرة على التغذية عن طريق الفم ونقص شديد في مستوى الصوديوم. استمرت عنده هاتان المشكلتان حتى تم تشخيصه بعد الولادة بخمسة اسابيع بنقص خلقي في افرازات الغدة الدرقية.

يقترح هذا التقرير آلية جديدة يمكن فيها تفسير زيادة السائل الأمنيوسي ناتجة عن نقص هرمون الغدة الدرقية، وليس عن طريق انسداد مجرى البلعوم الناتج عن تضخم الغدة الدرقية كما هو موصوف في تقارير اخرى. ثم مناقشة العلاقة بين عدم القدرة على التغذية بواسطة الفم وزيادة السائل الأمنيوسي في هذه الحالة، كما تم مراجعة حالات مشابهه منشورة حول موضوع دور الغدة الدرقية في السائل الأمنيوسي وتنظيم املاح الصوديوم في الجسم.

يدعو هذا التقرير الى ابحاث مستقبلية تساعد في فهم دور هرمون الغدة الدرقية في تنظيم املاح الصوديوم وانتاج البول في جسم الجنين.

الكلمات الدالة: زيادة السائل الأمنيوسي، نقص خلقي لهرمونات الغدة الدرقية، نقص شديد في مستوى أملاح الصوديوم.